CLAIMPTO

WNP

1. (Previously Presented) A chimeric live, infectious, attenuated virus, comprising:

a yellow fever virus in which the nucleotide sequence encoding a prM-E protein is either deleted, truncated, or mutated so that functional yellow fever virus prM-E protein is not expressed, and

integrated into the genome of said yellow fever virus, a nucleotide sequence encoding a prM-E protein of a second, different flavivirus, so that said prM-E protein of said second flavivirus is expressed, wherein the capsid protein of said chimeric virus is from yellow fever virus.

2. (Original) The chimeric virus of claim 1, wherein said second flavivirus is a Japanese Encephalitis (JE) virus.

6. (Original) The chimeric virus of claim 1, wherein the nucleotide sequence encoding the prM-E protein of said second, different flavivirus replaces the nucleotide sequence encoding the prM-E protein of said yellow fever virus.

7. (Original) The chimeric virus of claim 1, wherein said nucleotide sequence encoding said prM-E protein of said second, different flavivirus comprises a mutation that prevents prM cleavage to produce M protein.

8. (Currently Amended) The chimeric virus of claim 1, wherein the NS2B-3 protease recognition site and the signal sequences and cleavage sites at the C/prM and E/NS1 junctions are maintained in construction of said chimeric virus ~~flavivirus~~.

30. (New) The chimeric virus of claim 1, wherein said second flavivirus is a Murray Valley Encephalitis virus.

31. (New) The chimeric virus of claim 1, wherein said second flavivirus is a St. Louis Encephalitis virus.

32. (New) The chimeric virus of claim 1, wherein said second flavivirus is a West Nile virus.

33. (New) The chimeric virus of claim 1, wherein said second flavivirus is a Tick-borne Encephalitis virus.

34. (New) The chimeric virus of claim 1, wherein the signal sequence at the C/prM junction is maintained in construction of said chimeric virus.

9. (Previously Presented) A method of preventing or treating Japanese encephalitis virus infection in a patient, said method comprising administering to said patient a chimeric, live, infectious, attenuated virus comprising:

a yellow fever virus in which the nucleotide sequence encoding a prM-E protein is either deleted, truncated, or mutated so that functional yellow fever virus prM-E protein is not expressed, and

integrated into the genome of said yellow fever virus, a nucleotide sequence encoding a prM-E protein of Japanese encephalitis virus strain SA-14-14-2 or Japanese encephalitis virus strain Nakayama, wherein the capsid protein of said chimeric virus is from yellow fever virus.

14. (Previously Presented) The method of claim 9, wherein the nucleotide sequence encoding the prM-E protein of said Japanese encephalitis virus replaces the nucleotide sequence

encoding the prM-E protein of said yellow fever virus.

15. (Previously Presented) The method of claim 9, wherein said nucleotide sequence encoding said prM-E protein of said Japanese encephalitis virus comprises a mutation that prevents prM cleavage to produce M protein.

16. (Currently Amended) The method of claim 9, wherein the NS2B-3 protease recognition site and the signal sequences and cleavage sites at the C/prM and E/NS1 junctions are maintained in construction of said chimeric virus ~~flavivirus~~.